

affects the results of the ordinary standard tests for arsenic when the tests are carried out, as usual, in ordinary glass containers.

In the Gutzeit test, for example, hydrochloric or sulphuric acid, in reacting with the sodium fluorid mixture permits of the formation of hydrofluoric acid, which, in turn, attacks the wall of the glass container, dissolving sufficient quantities of the glass to permit of positive reactions for arsenic from the arsenic contained in the glass itself. Of course, with a prolongation of the time interval beyond the two-hour period ordinarily followed in standard procedure, the results obtained would indicate greater quantities of arsenic, for obvious reasons. Recheck analyses, in which the reactions were carried out in paraffin-lined containers, gave consistently negative results for arsenic. The only logical conclusion that can be drawn, therefore, as to the source of the arsenic in the mixture (as revealed in the laboratory studies executed during the field investigation by the Department of Public Health, the Coroner's Office, and the Food and Drug Administration of the United States Department of Agriculture) must consider that the varying quantities of arsenic found came, not from the mixture of sodium bicarbonate and sodium fluorid or commercial sodium fluorid, but from the glass containers in which the tests were carried out. It should be emphasized also that the time interval used in the test, as well as the quality of the glass containers themselves, are of considerable import in this respect.

#### SUMMARY

1. A mixture of sodium bicarbonate and sodium fluorid, sold in bulk as sodium bicarbonate or baking soda, was responsible for poisoning in twenty-one reported instances, three of which terminated in death.

2. Samples submitted by those made ill and by members of the families of the deceased, and those obtained by the inspectors of the Department of Public Health from the various sources involved (except those from intact barrels of the original product) consistently showed arsenic and fluorin on qualitative chemical analysis; on quantitative analysis these same samples were found to contain varying proportions of sodium bicarbonate and sodium fluorid, demonstrating a heterogeneous, "spotty," or "pocket" distribution.

3. The epidemiologic picture, at first confused by the positive reactions indicating the presence of arsenic in the specimens of "baking soda" (sodium bicarbonate-sodium fluorid mixtures) and of commercial sodium fluorid, was clarified through further study, which brought out the fact that hydrofluoric acid, found through the reaction of hydrochloric or sulphuric acid with the sodium fluorid of the mixture, dissolved the glass of the containers and released arsenic therefrom in sufficient quantities to give positive reactions for arsenic. Recheck tests in paraffin-lined glass containers gave consistently negative results for arsenic.<sup>1</sup>

4. It should be emphasized that there is no danger from sodium bicarbonate of standard

brands, sold in good condition, in the original package.

5. The incident should awaken interest in the real and potential hazards existing in the salvage of foods and drugs, and the need for effective legislation which will require and provide adequate official supervision and regulation over all persons, firms and corporations dealing in foods and drugs.

6. This incident, as also those reported by Simpson and his coworkers,<sup>2</sup> should serve as a warning and give impetus to the medical profession as well as local, state and federal authorities and officials, to secure adequate control over the labeling, manufacture, distribution, sale and use of insecticides, particularly those containing fluorin.

#### IN CONCLUSION

Fluorid poisoning, as a result of the ingestion of quantities smaller than those heretofore considered toxic for humans, is a very real hazard to the public health, even as a cause of death, in addition to the effects of the mottling of teeth, as reported from Arizona and other points,<sup>3</sup> and gastro-intestinal irritation. The control of traffic in insecticides should receive attention as a medical problem, rather than simply an agricultural and entomologic problem, affecting the personal and public health.

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### ACUTE FLUORIN POISONING\*

WITH REPORT OF FIVE CASES

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DISCUSSION by P. J. Hanslik, M.D., San Francisco;  
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**O**CCASIONALLY, in any year, a city the size of San Francisco is turned into a biological experimental laboratory by an unfortunate accident. The tragic residue of such misfortune accumulates in the civil courts of the city, while the scientific data is collected and compiled at the city morgue. As the result of a recent unfortunate circumstance in San Francisco, three deaths were recorded from the ingestion of almost pure sodium fluorid, and we wish to add to these three two more deaths which resulted from the voluntary ingestion of fluorid-containing insecticides with suicidal intent. All of these cases show consistent and similar anatomical changes which substantiate pathologic findings reported by others. They have an additional interest, however, in that they contribute to the knowledge of the mini-

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imum lethal dose of fluorid which will kill human individuals. It has been known for some years that reasonably small amounts of sodium fluorid or sodium fluorosilicate would cause death, and Marcovitch,<sup>1</sup> in 1928, discussed the acute toxicity of sodium fluorosilicate, reporting a fatal case where only half a teaspoonful of the substance had been ingested. Fatalities from less sodium fluorid than this are not available in the literature, but deaths from the substance have been reported by Sommelet,<sup>2</sup> Kockel and Zommerman,<sup>3</sup> Hickey,<sup>4</sup> McNally,<sup>5</sup> and Luhrig,<sup>6</sup> whose second patient had taken one-half teaspoonful of insecticide containing 99.6 per cent sodium fluorosilicate, while McNally reports death from ingestion of 4.5 to 5 grams of sodium fluorid. Much smaller doses than this, of course, will cause acute toxicity without death, and Kolipinski,<sup>7</sup> in 1866, pointed out that in human individuals any amount of sodium fluorid in excess of one-fourth grain (0.016 gram) is liable to cause nausea. Baldwin<sup>8</sup> reported that a quarter of a gram by mouth would cause nausea in two minutes, and Vallée,<sup>9</sup> in 1920, reported seven cases of poisoning by sodium fluorid where the amounts absorbed were reported as from 0.228 gram to 0.45 gram. Other reports seem to establish that the minimum toxic dose is somewhere around 0.05 of a gram, with the symptoms of gastralgia, nausea, vomiting, convulsions, paralysis of the motor center and salivation increasing to the minimum lethal dose, and that death would occur after the ingestion of four grams or perhaps a little less. The information on animals is somewhat more accurate. In 1867, Rubuteau<sup>10</sup> published data showing that when 0.5 of a gram was given to dogs by mouth, symptoms were produced, but he found that 0.25 of a gram by mouth produced no demonstrable changes. One gram intravenously caused serious symptoms, but was not fatal. In rabbits, 0.25 of a gram by mouth caused symptoms, and the same amount injected intravenously proved to be fatal. In 1894, Haidenhain<sup>11</sup> reported the fatal dose of fluorid for a dog to be 0.05 to 0.1 of a gram per kilogram of body weight when given intravenously. And Leake,<sup>12</sup> corroborating this finding, reported 87.5 milligrams per kilogram to be the minimum fatal dose intravenously in rabbits. Death occurred about forty minutes after injection of the minimum fatal dose. In continuing this work, Muehlberger,<sup>13</sup> in 1930, tested other fluorid salts than sodium fluorid, but with essentially the same findings. The symptoms he observed were salivation, diarrhea, tremor and, at times, terminal clonic and tonic convulsions. Animals receiving barely lethal or slightly smaller doses showed a persistent cachexia, and albuminuria was frequently present. In rabbits killed by sodium fluorid, the pathologic changes he noted were limited to the liver and kidney, the liver showing some congestion and hydropic degeneration, as well as some fatty changes in the cells. Schultz<sup>14</sup> found that lethal doses in experimental animals acted on the central nervous system, as well as upon the stomach and blood, producing a paralysis of the cord and brain, ending in stiffness, numbness, salivation, and gastric hemor-

rhage. The whole subject, in both acute and chronic forms, is presented in the excellent review of De Eds.<sup>15</sup>

In the first group of three cases to be reported, fluorid was taken in the form of sodium fluorid after being mistakenly sold for sodium bicarbonate and ingested with the same impression persisting. As far as can be determined, approximately half a teaspoonful, or three to four grams, was taken in these cases, with death following in from six and one-half to twelve hours.

#### REPORT OF CASES

##### 1. *Accidental Poisoning Group*

###### CASE 1.—Mrs. C. O., white, sixty.

At eight o'clock p. m. the subject was visiting a friend who had some gastric disturbance and suggested that she take a little soda for relief. The friend acquiesced, and took one-half of one teaspoonful (three grams) in water. Although feeling perfectly well, Mrs. O. also took half a teaspoonful (three grams) in water, to be sociable. They both noted an odd, rather bitter taste, and a few moments later both were nauseated. The friend immediately vomited her entire gastric content. Vomiting was attempted by Mrs. O., but was fruitless. There was a rather rapid prostration, a feverish feeling, and in fifteen minutes a diarrhea. Severe oral burning ensued and the tongue was sore. She remained with her friend, who had been able to vomit repeatedly, until midnight, when she went home. At this time, approximately four hours after ingestion, she was very weak, nauseated, and having a premonition of death requested an autopsy in the event of her demise. Her brother gave her some hot water and then retired. She died unobserved at approximately 2:30 o'clock the following morning, six and one-half hours after the initial symptoms and ingestion. The autopsy findings were consistent with the composite report, which follows. Chemical analysis showed fluorid and a trace of arsenic in the stomach, liver, and kidneys. Analysis of the material ingested showed 99 per cent pure sodium fluorid, and 250 parts per million of arsenic, the latter being regarded as a commercial impurity. Subsequent studies show the arsenic to have been dissolved out of pyrex glass of the container by the etching action of sodium fluorid solution.

###### CASE 2.—Mrs. B. S., white, fifty-three.

It was the subject's custom to take a little baking soda twice a week in water shortly after arising in the morning. At 7 a. m., the day of her death, she entered the kitchen shortly after dressing and took one-half of one teaspoonful of a substance which she had purchased in bulk the previous day, and supposed was baking soda. Her husband found her a few moments later sitting in a chair with perspiration beginning to appear on her forehead. She remarked immediately: "Leave that baking soda alone—it is poison." Inside of ten minutes there was extreme nausea and vomiting, with diarrhea following about fifteen minutes after ingestion. A doctor was summoned and arrived approximately twenty-five minutes after the material was taken. He found the patient pulseless and prostrated. She was put to bed, still vomiting, and was given hot water each time emesis occurred. The type of poison was not known and no antidote was given. There was no abdominal pain. In approximately an hour she was semi-comatose and began to have successive chills. Hot-water bags were placed about her, following which she relapsed into coma again, from which she would awake, vomiting. She grew progressively weaker and had a constant premonition of death. At 2 p. m. she felt cramps in her legs and had some clonic spasms. She was given an alcohol rub, which eased her somewhat. About 2:30 p. m. her whole body broke out in a rash. Weakness was increasing. She had a feeling that she was burning up inside. She begged for water, which was administered, and attempted to vomit at this time, but was unable to raise any fluid. The patient was extremely weakened

and muscular contraction was difficult. She was semi-conscious, however, and able to converse with her daughter. At 3 p. m., eight hours after ingestion of the poison, she died, apparently from a respiratory paralysis. The autopsy findings in this case were consistent with those which are subsequently described. Fluorid was not recovered from either her stomach or viscera. The material ingested was 84.5 per cent sodium fluorid.

CASE 3.—Mr. A. T., age eighty-six. Retired carpenter. Father of Mrs. S., Case 2.

The patient was found in bed about 2 p. m. by neighbors, who immediately summoned a doctor. Examination showed him to be salivating, frothing at the mouth and nose, practically pulseless, cold and comatose. He was transferred to the Central Emergency Hospital. By emesis in the sink, it was concluded that he had been vomiting. Because of this the stomach was not lavaged at the Emergency Hospital, but the patient was put to bed and kept warm. He improved, and at approximately five o'clock in the evening was able to talk. He gave a history of having eaten a large dinner the day before and, feeling distressed that evening, had taken baking soda. The following morning, upon awakening at seven o'clock, he felt sick and nauseated, and took approximately one-half teaspoonful more in water, the soda having been part of the package his daughter Mrs. S. purchased the previous day. This made him acutely ill and he vomited a number of times, which was followed by diarrhea. The vomiting and diarrhea continued the better part of the day. Later in the afternoon he noticed that his hands were growing weak and gradually becoming paralyzed. He felt very weak and had some pain across the lower part of his chest. Froth, which had been found exuding from his nose when first visited by his doctor, had disappeared, and at 5:30 p. m. his temperature was normal, blood pressure was normal, and pulse was 85. His hands and arms were paralyzed and there was some weakness of the lower limbs. He grew gradually weaker and died at 7:20 p. m. that day, approximately twelve hours after ingestion of the poison. Toxicologic examination of the stomach was negative for fluorid, and the presumption is that he had completely evacuated the substance by vomiting.

#### COMMENT

The clinical similarity of these cases is apparent, the only variation being rapidity of death after ingestion. The shortened interval in the first instance, we believe, is due to the inability to evacuate the stomach by vomiting, and this is substantiated to a great extent by the finding of fluorid in the stomach of the first individual, and by its absence in the stomach of the other two.

### 2. Suicidal Poisoning Group

The second group of two cases were the results of voluntary suicidal ingestion and, while no histories are, of course, available, it appears by the amount of insecticide remaining in either can that something less than an ounce of the material was taken. Both individuals were found dead in the morning, having died during the preceding night. Fluorid was recovered from the stomach in both instances.

#### PATHOLOGIC FINDINGS

The pathologic changes in all cases are consistent, and will be reported as a composite picture. Upon the initial incision one is struck by the extraordinary congestion and hemorrhagic infiltration of all of the organs, but particularly of the spleen and lungs. Throughout the pulmonary parenchyma, there is a dilatation of the vessels and congestion of the vascular channels, and blood oozes from the alveoli as the tissue is cut. Because of this infiltration the lungs are increased in weight, but they show no focal areas of necrosis or other damage. The heart varies with age

and physical influences, but shows, in addition, a moderate dilatation, loss of muscle tone, and a congestion of the vascular beds. The liver is quite yellow, but the edges are sharp and the color is uniform. There are no areas of necrosis or acute damage, but on section there is a cloudy swelling apparent throughout. The spleen is enlarged, acutely congested and engorged with erythrocytes. The lymphoid elements are not prominent. The pancreas appears grossly normal, but the kidneys show an acute congestion, are rather edematous and moderately swollen. The reproductive organs in both male and female show no pathologic changes. In the gastro-intestinal tract there is an acute congestion, and hyperemia of the gastric mucosa, as well as similar changes to a lesser degree in the rest of the bowel. There are small points of hemorrhage in the gastric mucosa, and the stomach contains blood-stained fluid.

Microscopic sections show an edema of the heart muscle, with some separation of the fibers and a little cloudy swelling of the cytoplasm. There is a general congestion of the circulatory channels, but no apparent endothelial damage. The lungs show the diffuse congestion described grossly with cells packed densely into the capillaries of the alveolar walls, and with fresh hemorrhage in the alveolar spaces. There is no acute focal necrosis and no evidence of inflammation other than the acute congestion. The liver shows a moderate fatty infiltration of the cells, with large droplets in the cytoplasm. The cytoplasm, in addition, is quite cloudy, and the cells are swollen. There is no degeneration or breaking up of the nuclei, however, and no changes about the portal areas. The spleen is congested with erythrocytes and the sinusoids are packed full of these cells. There is no apparent change in the white cell element, while the pulp and corpuscles are normal excepting for a moderate edema. The acinar cords of the pancreas are normal and the islets show no changes. The glomerular tufts of the kidneys are congested, and the capillary channels filled with erythrocytes. The glomerular capsules are clean, however, and the capsular spaces contain no exudate. There is a general cloudy swelling of the cells lining the tubules. The sections of the genital system are normal. Sections of the gastro-intestinal tract show an acute hyperemia and congestion of the circulatory channels. There is a little blood on the surface of the mucosa, and some of the mucosal cells are cloudy and beginning to degenerate. Changes in the central nervous system are limited to edema and a congestion of the vascular channels.

#### COMMENT

These findings are consistent with the pathologic observations in the previous cases of fluorid poisoning which have been reported by others. The clinical symptoms, too, substantiate published reports excepting the rash which appeared currently in Case 2. This, we feel, was not a toxic rash, as has been suggested, but a multiplicity of petechial hemorrhages following the chemical action of the fluorid ingested. As Kobert suggests in his "Lehrbuch der Intoxikationen," the

coagulation of the blood is abolished because of the binding of the calcium with fluorin, forming the insoluble calcium salt. While this exact mechanism may be subject to some question, there is no question that sodium fluorid is an active and efficient anti-coagulant, and such a property was probably operative in the individual. The hemorrhages were the same cutaneous petechial type that one would see in any other hemorrhagic diathesis, whether chemical or due to physiologic changes in the preformed clotting elements of the blood. The consistent bloody fluid found in the stomach is also probably partly due to this anti-coagulant factor.

The mechanism of fluorid toxicity appears to be most logically on the basis of a combination of a fluorid ion on calcium metabolism, as suggested by Kobert and others, as well as from its inhibitory action on lipase and other enzymes as reported by Amberg and Loevenhart.<sup>16</sup> According to Embden and Lange,<sup>17</sup> and Embden and Hentschel,<sup>18</sup> the presence of increased amounts of fluorid ion interferes with equilibrium between lactocidogen, lactic acid and phosphoric acid in the blood stream. This, as De Eds<sup>15</sup> suggests, may contribute, in interfering with the physiology of normal muscle contraction, to the extreme weakness experienced by these patients. From pathologic observations it is apparent that the salt acts as an acute protoplasmic poison, and the cloudy swelling and early cytoplasmic damage which is seen in all of these cases is the result of its influence.

While none of these cases of either accidental or voluntary intoxication were apprehended early enough to attempt the administration of a remedy, it has been suggested by Vallée, and Stanton and Kahn that administration of a soluble calcium salt would, to a great degree, bind the mobile fluorid ion and result in detoxification. Large amounts of milk have been suggested and used with some benefit, due probably to the calcium effect, while Stanton and Kahn<sup>19</sup> report a case that recovered after a lethal dose of sodium fluorid, which was treated by massive gastric lavage, lime water and calcium chlorid.

#### CONCLUSIONS\*

1. Five cases of acute fluorid poisoning are reported which corroborate the clinical and pathologic findings of previous reports.

2. Terminal cutaneous petechial hemorrhages are observed, and their presence attributed to the anti-coagulant action of fluorin.

3. The mechanism of fluorid toxicity is probably due to a combination of formation of an insoluble calcium salt, and by the interference of the action of enzymes and lactic acid metabolism.

4. Three grams is a sufficient quantity to cause death in man.

5. Gastric lavage and subsequent stomach washing with lime water, milk, or other calcium salts, are at present the most proper treatment.

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#### DISCUSSION OF PAPERS OF DRS. GEIGER AND CARR

P. J. HANZLIK, M. D. (Stanford University School of Medicine, San Francisco).—Doctor Geiger's paper covers the problems of acute and chronic fluorid poisoning so well that I can add but little more than approval. Doctor Geiger suggests the proper remedy for avoidance of such a shocking occurrence as happened in San Francisco, and the public is clearly entitled to that protection. No one today, who fully understands the phenomena of chronic intoxication, would disagree with the contention that insecticides and spray residues constitute a public hazard of large proportions. Minute quantities of these poisons act so insidiously and slowly that they may actually be the etiologic factors in some common disorders, the symptoms being much alike. Fluorid, in any form, is one of the most dangerous insecticides and contaminants of natural foods and waters. So-called "mottled enamel" or bleaching of growing teeth in animals occurs after taking in food something like only fourteen parts per million of fluorid, and in children one part per million. There are probably other systemic abnormalities caused by prolonged ingestion of fluorid. The action is apparently not a simple matter of decalcification, because calcium fluorid produces the same effects. What is fundamentally involved in fluorosis is as yet undetermined, although some intrinsic impairment of protoplasm and enzymes appears plausible, which merely emphasizes the insidiousness of this intoxication. The complete elimination of fluorin as an insecticide, and use of soils and waters where it is a contaminant is the only remedy at present. Constructive work on insecticides should concern itself with the development of volatile organic substances which leave no residues, in order to eliminate all hazards to health. This paper again indicates Doctor Geiger's alertness and constructive attitude on all matters pertaining to the health of the people.

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P. J. HANZLIK, M. D. (Stanford University School of Medicine, San Francisco).—Doctor Carr's summary brings up to date practically all that is known about acute fluorid poisoning. The disionization of calcium in the blood and tissues is undoubtedly responsible for the symptoms and tissue changes of the acute poisoning, and the more soluble the fluorid the more rapid the onset and more violent

the symptoms, as occurred in these cases. Chronic poisoning is, of course, different fundamentally, since calcium fluorid and insoluble fluosilicates produce the intoxication quite readily. Calcium is the choice antidote for acute poisoning and should be used locally and systemically, the gluconate being better tolerated than the chlorid, both intramuscularly and intravenously. Charcoal can be profitably given in liberal quantities and left in the stomach after the lavage is completed. Tremors or convulsions, if present, can be controlled with barbitol. According to Doctor Carr, the total fatal dose for a man is much smaller than has been frequently supposed. In the presence of food, recovery can probably result from larger quantities.

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C. D. LEAKE, Ph.D. (University of California Medical School, San Francisco).—As Professor P. J. Hanzlik of Stanford University has remarked in other discussions of reports by Doctor Geiger and the members of the staff of the Department of Public Health of San Francisco, the citizens and medical profession of the city may be extremely grateful for their splendid work. Doctor Geiger has developed a technique of frankness and honesty in his relation with the public and the profession which is almost unique, but which has been responsible for great and significant improvement in public health matters in San Francisco. The promptness with which Doctor Geiger and Doctor Carr are reporting to their scientific colleagues the essential facts in the recent fluorin poisonings in San Francisco is eloquent testimony of their desire to cooperate fully with their colleagues in every affair concerning public health. The difficulty of arriving at an accurate picture of the unfortunate situation is graphically described by Doctor Geiger. There is no doubt but that this incident will serve to bring about much more satisfactory public health control of hitherto unregulated food and drug dealers. It will also serve to bring to the city chemist improved apparatus and facilities which he has for so long needed.

Doctor Carr's report is an excellent review of the essential features in acute fluorin poisoning. The widespread use of various fluorids in pest control makes it necessary that all physicians be aware of the possibility of acute poisoning from these substances. The protection of water supplies to prevent chronic fluorin intoxication is a public health matter, and physicians must also be familiar with the symptoms of such poisoning. The review made by De Eds (*Medicine*, 12:1, 1933) is the best available on this matter.

It is interesting that the amounts of sodium fluorid taken by the individuals mentioned in Doctor Geiger's and Doctor Carr's reports is in the range of the minimal fatal dose that we found in animals. We found that 87 milligrams per kilogram would kill about half of the animals into which it was intravenously injected, and later we found that approximately this same dosage given by mouth was also in the minimal toxic range. This latter work was not reported. It was our opinion that the chief factor in acute fluorid poisoning is calcium precipitation. However, we brought forward definite evidence to indicate inhibition of enzyme action throughout the body (*American Journal of Physiology*, 90:426, 1929). We had previously found (*American Journal of Physiology*, 76: 234, 1926) that sodium fluorid by mouth is extremely irritating to the stomach mucosa, rapidly producing marked congestion and erosion in high concentrations. Although the weights of the patient referred to are not given, they took amounts of sodium fluorid apparently around three grams or more. Since there were two elderly women who died and one old man, it may be assumed that their weights were not very great and that they ingested and absorbed amounts of sodium fluorid in the range of 60 to 90 milligrams per kilogram.

The fluorids constitute a definite public health hazard, both from the standpoint of the accidental or suicidal ingestion of insect powders or chronic poisoning from contaminated water supplies. These reports by Doctor Geiger and Doctor Carr comprise an excellent survey of these dangers, and of the necessity of attempting to control them.

## PSYCHIATRY AND THE LAW\*

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DISCUSSION by *Walter Rapaport, M.D., Imola; J. M. Scanland, M.D., Agnew; Margaret H. Smyth, M.D., Stockton.*

MUCH confusion has existed in the minds of laymen as well as physicians, and also in the minds of attorneys regarding the use of the term "insanity." Many maintain that the term is purely medical, while others as seriously contend that it is a legal contribution, both as to name and as to conception. Glueck states that the specific term "insanity" seems first to have been introduced into the law by medical experts, finally superseding such terms as madness, lunacy and mental unsoundness; which were borrowed from the very imperfect psychiatric knowledge of the day. The word "insanity" seems to have been quite generally used by commentators on the law in early times, and it is probable that all such terms were popularly used until they were taken over by the first systematic writers both in law and in psychiatry, and applied to this specialized field.

### RULES OF LAW GOVERNING INSANITY AS A DEFENSE TO CRIME

The rules of law governing insanity as a defense to crime are vague and confused; clearly unsound in that they are based upon notions of the mental disorder discredited by medical science. Owing to the careless use of language by judges and others in the law, insanity has become synonymous with criminal irresponsibility, by reason of a certain type and degree of mental disorder which was noted far in advance by means of artificial tests of criminal responsibility. Because the law has preempted the term "insanity" and specialized it, judges frequently employ the term in the sense of irresponsibility, while psychiatrists seek other terms to distinguish the mentally-ill without reference to criminal responsibility. This practice has resulted in judges on the bench frequently attempting to distinguish between "legal insanity" and "medical insanity." This subject has been under discussion by English and American courts, legislators, and legal writers for more than a century. As long ago as 1800 the brilliant Lord Erskine, in his argument as counsel in the trial of Hadfield, attempted to lay down a universal test of responsibility in cases where the defendant suffered from a mental disease. His effort and the efforts of others have yet failed to attain clarity and uniformity; and even to this date, no universal test of responsibility has been generally adopted. There are many reasons for this; but for a branch of learning which has as a specialized objective largely the definition of words and their making, the law is strangely lax in its use of the term. Unfortunately, the word has no technical meaning either in law or in medicine, and is used by the courts and by legislators ordinarily to convey either one or two meanings:

\* Read before the Neuropsychiatry Section of the California Medical Association at the sixty-fourth annual session, Yosemite National Park, May 13-16, 1935.